ON THE PHYSIOLOGY OF ASPHYXIA

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ANÆSTHETIC ACTION OF PURE NITROGEN

BY

GEORGE JOHNSON, M.D., F.R.S.

CONSULTING PHYSICIAN TO KING'S COLLEGE HOSPITAL, EMERITUS PROFESSOR OF CLINICAL MEDICINE,
PHYSICIAN EXTRAORDINARY TO H.M. THE QUEEN

Reprinted from "THE LANCET," April 4 and 11, 1891

With the Author's kind regards.

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Prologue.

The following paper was read (in abstract) and discussed at a meeting of the Royal Society on Feb. 5th, 1891. In now submitting it to the readers of The Lancet, I have interpolated a few short paragraphs and notes which are distinguished from the rest of the paper by being included within brackets. The subjects here discussed are such as should excite the interest not only of physiologists, but of all who are engaged in the practice of medicine and surgery. It is impossible to correctly interpret the character of the pulse in various morbid conditions without a clear understanding of the influence of the arterioles, both pulmonary and systemic, upon the circulation of the blood. The phenomena of asphyxia from exclusion of atmospheric air, and the results of the inhalation of pure nitrogen and other azotic gases, afford most instructive illustrations of the regulating and controlling influence of the arterioles upon the circulation. These interesting and instructive phenomena it is the main object of this communication to describe and explain, in accordance with the established facts and principles of physiology.

In a recently published Essay on Asphyxia (Apnœa) I have endeavoured to prove that those physiologists who

accepted the doctrine of Alison, Reid, and Erichsen—that the immediate cause of death from what is commonly called asphyxia is the arrest of the pulmonary circulation—were right; and that the more modern doctrine, which assumes that the circulation is finally arrested in consequence of the muscular walls of the heart being paralysed by the circulation of unaerated blood through the tissues, is erroneous. Since the publication of the essay in question I have thought it desirable to obtain additional evidence in support of the theory which I first published twenty-two years ago—viz., that the arrest of the circulation in the final stage of asphyxia (apnœa) is caused by the contraction of the muscular-walled pulmonary arterioles.[1]

I have to express my obligation to my friend Mr. Charles James Martin, M.B., B.Sc., Demonstrator of Physiology, King's College, London, for the time and labour which he has bestowed upon the skilful performance of numerous and varied experiments, the results of which will, I venture to say, throw much light upon the complex phenomena of asphyxia. It is right to mention that Mr. Martin is not responsible for my interpretation of the results of his experiments.

And here it may be well to state that all these experiments were performed on animals under the influence of anæsthetics—usually morphia, subcutaneously injected, and in some cases with the addition of chloroform—anæsthetics which, while they entirely prevented suffering on the part of the animals, did not interfere with the physiological results of the experiments. Every animal was finally killed by deprivation of air. Animals—rabbits, cats, and in a few cases dogs—were asphyxiated either by ligature of the trachea, by the paralysing influence of curare, or by causing them to inhale a gas containing no free oxygen—viz., nitrous oxide, pure nitrogen, hydrogen, and carbonic acid gas. In all these experiments reinspiration of the gases was avoided by allowing the expired gases

[1] Med. Chir. Trans., vol. li., 1868, p. 68.

to escape through a T-tube fixed in the trachea. During the performance of the experiments in most cases the chest and pericardium of the animals were opened, so that the relative degree of fulness of the heart's cavities might be readily observed. In all these experiments the results as regards the distension of the heart's cavities were essentially the same, no matter whether the air was simply excluded, or whether an azotic gas (i.e., a gas not in itself poisonous, yet unable to support life) was substituted for atmospheric air, the only difference being that in the case of inhalation of an azotic gas the phenomena are more rapid in their occurrence, in consequence of the more speedy displacement of oxygen from the lungs.

The principal changes in the heart's cavities were as follows:—1. Distension of the left cavities of the heart. 2. Enormous distension of the right cavities, with diminished distension of the left; the distension of the right cavities being so great that the enlarged heart actually bulged out between the split sternum of the animal; the lungs at the same time being pale, bloodless, and collapsed to an extreme degree. The circulation was apparently brought to a standstill by the inability of the right cavities to empty themselves in consequence of opposition in front.

That the arrest of the circulation could not be due to paralysis of the heart's walls by the circulation of venous blood through its tissue is, I think, conclusively proved by the following experiment amongst others.

Into the trachea of a small dog, prepared with the chest and pericardium opened, and kept alive by artificial respiration, as above described, a glass T-tube was introduced, through which pure nitrous oxide gas was passed into the lungs, whilst the expired gases escaped into the air. As usual, first the left, then the right cavities became distended, and in one minute the heart's action had nearly ceased, with over-distension of the right side. Then, without loss of time, inhalation of nitrous oxide, *impregnated with the vapour of nitrite of amyl*, was substituted for the pure N_2O, by means of a two-way stopcock, and the

result was that almost immediately the distension of the right cavities began to subside, and in two minutes they had nearly regained their normal size.

It was found that after the inhalation of the nitrite of amyl the lungs had assumed a peculiar dirty yellowish tinge, and artificial respiration of air failed to restore life. It is well known that nitrites convert hæmoglobin into methæmoglobin, as a result of which the oxidation of the tissues is hindered. It appears to me that the only possible explanation of these phenomena is that the circulation having been arrested by contraction of the arterioles, was for a time restored by the paralysing influence of the nitrite of amyl upon those vessels, atmospheric air being all the time strictly excluded.[1]

Additional evidence of the influence of the arterioles in arresting the circulation during the progress of asphyxia is derived from the fact that a sufficient dose of such agents as are known to paralyse the arterioles—e.g., curare and atropine—prevents over-distension of the heart's cavities, and considerably prolongs the life of the animal.

The following experiment was performed upon a rabbit previously narcotised by morphia. The chest and pericardium being opened and the animal kept alive by artificial respiration, nitrous oxide was then substituted for air. In half a minute there was distension of the left cavities, followed in one minute by distension of the right. In one minute and a half enormous distension of the right cavities was observed, with convulsions. In two minutes and a half the heart stopped. Artificial respiration with air was now resumed. The heart immediately recommenced to beat, and its distension was removed. The heart's cavities were of the normal size and fulness in seventy seconds after

[1] [During the discussion of my paper at the Royal Society an eminent physiologist said, truly enough, that nitrite of amyl relaxes the systemic as well as the pulmonary arterioles; but that the disproportionate distension of the right cavities in this experiment was the result of contraction of the *pulmonary* arterioles can, I think, scarcely be doubted.

the readmission of air into the lungs. Then a decigramme of curare was slowly introduced into the jugular vein, the introduction occupying five minutes. Three minutes after the completion of the curare injection inhalation of N_2O was resumed and continued. The result was that the heart continued to beat slowly and feebly, that there was no distension of its cavities, that there were occasional slow contractions ten minutes after the commencement of the N_2O inhalation, and even fifteen minutes afterwards the heart was still flickering. In this experiment the dose of curare had been sufficient to paralyse the arterioles.

Into the peritoneal cavity of a cat a decigramme of curare was injected, the animal being kept alive by artificial respiration. When the respiration was suspended, the blood pressure in the carotid immediately began to fall, and artificial respiration had to be resumed to prevent the complete arrest of the circulation by cardiac paralysis. The same phenomena occurred more than once after the respiration had been suspended. At length, after an interval of nearly an hour, suspension of respiration was followed by a rise of blood pressure in the carotid; and the heart having been exposed, there was seen to occur the usual distension, first of the left cavities, then of the right. In this experiment the excessive dose of curare had paralysed the arterioles and weakened the heart. The suspension of respiration still further weakened the heart, and caused an immediate fall of blood pressure. After a time a portion of the curare was eliminated, probably by the kidneys, a considerable amount of urine having been passed upon the operating table, and then the usual results of a moderate dose of curare were observed.

The following experiment was performed upon a cat, previously narcotised by morphia and chloroform. A dose of curare was injected, sufficient to paralyse the voluntary muscles but not the arterioles. A manometer tube, connected with the kymograph, was introduced into one carotid. Suspension of the respiration was followed by the usual rise and subsequent fall of systemic arterial pressure. The cir-

culation having been restored by artificial respiration, sulphate of atropine was injected into the jugular vein; after which the arrest of the breathing produced no rise of pressure in the carotid, and when the sternum was split and the heart exposed, no distension of its cavities was observed.

The three experiments above described suffice to prove that the distension of the heart's cavities and the arrest of the circulation during the progress of asphyxia are the result of arterial contraction, and not of paralysis of the heart's walls by the circulation of venous blood through its tissues.

It has been suggested that the distension first of the left, then of the right side of the heart, is the result solely of *systemic* arterial contraction, the impediment acting backwards from the left side of the heart through the lungs to the right cavities and the systemic veins. One main objection to this theory is the fact, I think generally admitted, that when the chest is opened immediately after death from asphyxia, the lungs are found extremely pale, from anæmia of their minute vessels, and in a corresponding degree collapsed. Surely a backward pressure from the left side of the heart, sufficient to greatly distend the right cavities, must of necessity involve engorgement of the pulmonary capillaries. That there is a certain amount of backward pressure from the primary distension of the left side, extending as far as the *pulmonary veins*, would seem to be proved by observations made by Mr. Martin, to the effect that a manometer in a branch of a pulmonary vein indicates an early and continuous increase of pressure during the progress of asphyxia. But that this backward pressure does not cause the distension of the right side of the heart is shown by the fact that in the last stage of asphyxia, while the right cavities are in a state of extreme distension, the left are, as a rule, flaccid and comparatively empty, the lungs, as before mentioned, being very anæmic and collapsed to an extreme degree.

[Moreover, if the distension of the right cavities of the

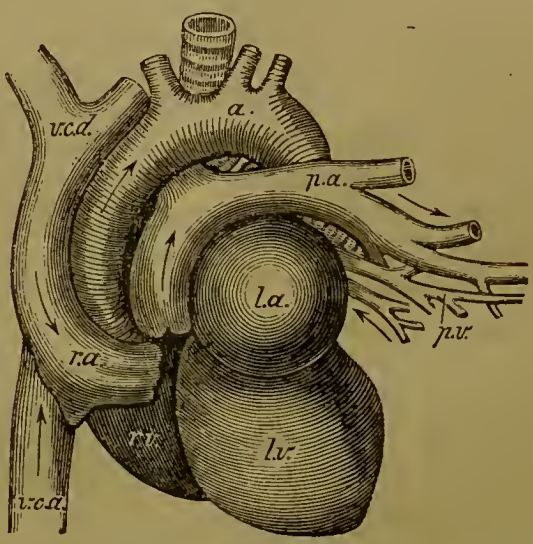
heart were a result of backward pressure from the left, not only would the pulmonary capillaries be engorged, but the pressure in the pulmonary veins would be greater than that in the pulmonary artery, the fact being that the venous pressure is so much less than the arterial, that it can be measured only by a water manometer.]

A most complete demonstration of the condition of the heart's cavities in the different stages of asphyxia was afforded by some experiments performed by Dr. Rutherford, now Professor of the Institutes of Medicine at the University of Edinburgh, which I had the privilege of witnessing when he was my colleague at King's College in the year 1873. The following is a brief description of Dr. Rutherford's experiments.

Into the trachea of a large dog, previously anæsthetised by chloroform, a tube was tied, and connected with a bellows for the performance of artificial respiration. The voluntary muscles were then paralysed by the injection of a moderate dose of curare, and the animal was kept alive by artificial respiration. The sternum and portions of the ribs were removed, and the pericardium was opened, so as to expose the anterior surface of the heart. One common carotid artery was divided, and a dynamometer tube connected with a mercurial kymograph was introduced into the proximal end. Artificial respiration was now suspended, and immediately the colour of the left auricle changed from crimson to purple, the dark venous blood showing through the thin walls of the auricle, while the kymograph indicated a continuous rise of pressure in the systemic arteries, the variations of arterial pressure being registered by a pen on a revolving cylinder. After the increase of pressure had continued for about a minute the *left* cavities of the heart were much distended, the auricle in particular becoming expanded into a tense globular ball with a smooth surface. (See Fig. 1.) In the next period the pressure in the systemic arteries began to fall, and about the same time the right cavities of the heart, which had hitherto remained of the normal size and form,

became distended, while the distension of the left rapidly subsided. Meanwhile the right cavities became more and more distended, and now the *right* auricle assumed the appearance of a tense globular ball, while the left auricle had become flaccid and nearly empty. The right ventricle also became so distended that it projected above the level

FIG. 1.

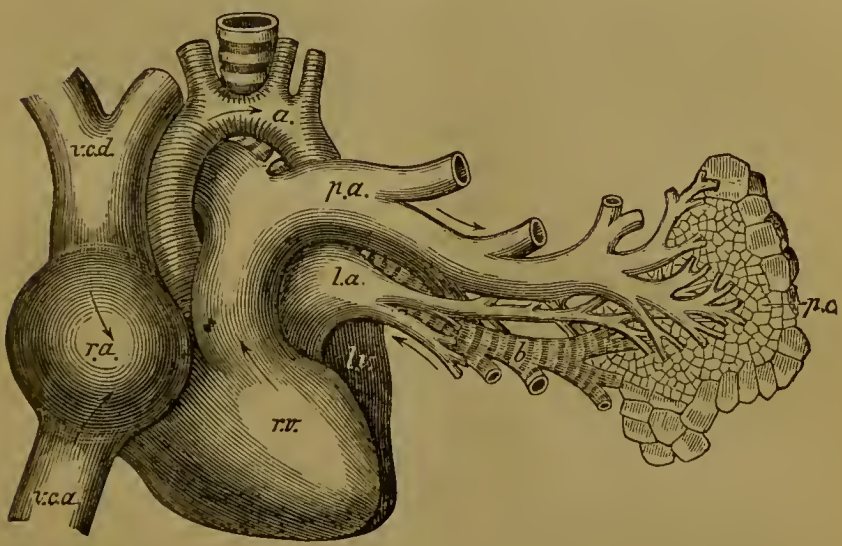


Represents the distension of the left cavities of the heart and aorta in the first stage of apnœa (asphyxia). *l.a.*, Left auricle; *l.v.*, left ventricle. Both greatly distended, the former like a smooth india-rubber ball. *a.*, Aorta distended; *p.a.*, pulmonary artery; *p.v.*, pulmonary vein; *r.a.*, right auricle; *r.v.*, right ventricle; *v.c.d.*, descending vena cava; *v.c.a.*, ascending vena cava. The right cavities of the heart, the pulmonary artery, and the systemic veins are in a state of normal fulness. The right ventricle is partly overlapped by the distended left.

of the left. (See Fig. 2.) This was the condition of the heart's cavities when the animal died by the final arrest of the circulation through the lungs; but more than once, when the circulation was nearly at a standstill, artificial respiration was resumed, and then all the phenomena rapidly changed. The blood, which had accumulated in

the pulmonary artery and in the right side of the heart, at once passed freely through the lungs, the distension of the right cavities subsided, and the systemic arterial pressure became first excessive, while the blood was partly venous, and then normal, when the blood became thoroughly oxygenised, and its passage through the terminal systemic arterioles was therefore no longer abnormally resisted.

FIG. 2.



Represents the distension of the right cavities of the heart, of the pulmonary artery, and the large systemic veins in the final stage of apnœa (asphyxia). The letters have the same significance as in Fig. 1. In addition, *p.c.* indicates the anæmic condition of the pulmonary capillaries; *b.*, left bronchus. The right auricle and ventricle and the pulmonary artery are fully distended, the auricle having the form and smoothness of a distended ball, while the left cavities of the heart and the aorta are collapsed and nearly empty.

[The following is the explanation of the facts revealed by Dr. Rutherford's most instructive experiment:—Artificial respiration being suspended, unaerated dark blood at first passed freely to the left side of the heart and to the systemic arteries. Arrived there, either by its direct stimulation of the muscular arterioles, or by a reflex influence through

the vaso-motor nerves and centre, the muscular arterioles[1] are excited to contract, and by this action of the vaso-constrictors the blood pressure in the arterial trunks is increased, while the left cavities of the heart become distended and dilated, as seen in Fig. 1. The circulation through the systemic arterioles is impeded and lessened, but not entirely arrested ; some black blood passes through the capillaries, and this venous blood, becoming more and more entirely deoxidised, reaches the right side of the heart, and the pulmonary vessels, and coming in contact with the pulmonary arterioles, it excites in them the same contraction and resistance as had before occurred in the systemic vessels. The resistance offered by the pulmonary vaso-constrictors, while on the one hand it tends to empty the left side of the heart and to lessen the blood pressure in the systemic arteries, on the other it causes that great distension of the right cavities and of the systemic veins which is invariably found when the chest is opened immediately after death from asphyxia, and which in such experiments as Dr. Rutherford's are plainly seen to occur during the lifetime of the animal. (See Fig. 2)] The relative amount of blood on the two sides of the heart is not an exact measure of the degree of pulmonary obstruction which has existed during the last moments of life, for the reason that the impairment of the contractile power of the left heart by anæmia and venous blood appears to vary in different cases ; but I may here mention the results of the simple experiment of ligaturing the trachea of a dog which has been twice performed in my presence ; the chest having been opened and the amount of blood in the two sides of the heart measured immediately after the animal had ceased to struggle.

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1 It has been shown in Ludwig's laboratory that the arteries of an organ which has been withdrawn from all nervous influence contract when blood overloaded with carbonic acid flows through them. See Conheim's Lectures on General Pathology : New Sydenham Society's Transactions, p. 112

In one dog, weighing 19¼ lb., 2 oz. of blood gushed from the distended right cavities, while 2½ drachms of blood flowed slowly from the comparatively empty and flaccid left side. In a dog half the size of the above, the experiment having been performed by Mr. Martin, the right auricle was found distended, the left empty; the right side of the heart contained 5½ drachms of blood, the left a quarter of a drachm.[1]

The explanation which I have before given of these facts appears to me to be the true one—viz., that during the later stages of asphyxia, when the blood has become entirely deoxidised, the pulmonary arterioles contract and cause the extreme distension of the right cavities, with anæmia of the pulmonary capillaries and a corresponding defective blood-supply to the left cavities of the heart.

[The extreme distension of the right cavities of the heart while the left are flaccid and comparatively empty, the minute tissue of the lungs being pale and bloodless, clearly indicates an impediment to the flow of blood between the right ventricle and the pulmonary capillaries. Such an impediment is explained by the contraction of the pulmonary arterioles—the vaso-constrictors of the lungs. It appears to me that the phenomena of asphyxia afford a more complete proof of the powerful action of the pulmonary vaso-constrictors than can be derived from any kymographic tracing.]

I venture to suggest the following explanation of the increase of blood pressure in the pulmonary veins, which has been observed by Mr. Martin and other experimenters during the successive stages of asphyxia. In the first stage, when, with high systemic arterial tension, the left cavities of the heart are distended (Fig. 1), there must be a backward pressure in the pulmonary veins, extending, perhaps, as far

[1] The results of these experiments suffice to prove the error of those physiologists who maintain that the comparative emptiness of the left cavities after death from asphyxia is the result of rigor mortis.

as the pulmonary arteries.[1] It is probable, however, that a concurring, if not the main, cause of the pulmonary venous pressure which continues and sometimes increases in the second stage of asphyxia, when the systemic arterial tension and the distension of the left cavities have passed away, is the compression of the pulmonary veins by the extreme collapse of the lung which occurs when the chest is opened in order to introduce a manometer into one of the pulmonary vessel trunks.[2] It is an acknowledged fact that the comparatively slight compression of the pulmonary veins which occurs towards the end of an ordinary expiration lessens the flow of blood into the left side of the heart. The degree of impediment to the pulmonary venous current which is thus occasioned must, however, be very much less than that which results from compression of the veins by the elastic force of the collapsed lung when an opening has been made in the wall of the chest.

Lastly, it is probable that yet another hindrance to the onward flow of blood in the pulmonary veins in the second stage of asphyxia is the impairment in the suction power of the left auricle, partly by anæmia of the cardiac tissues consequent on contraction of the arterioles, both pulmonary and systemic, the coronary included,[3] and partly by the fact that the small amount of blood with which the heart is supplied is more or less completely deoxidised.

[The facts and considerations here stated appear to me to explain the circumstance that, whereas the immediate

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[1] This backward pressure will account for the fact mentioned by Drs. Bradford and Dean (Proc. Roy. Soc., vol. xliv., No. 277), that in the early stage of asphyxia there is a synchronous rise of pressure in the systemic and pulmonary arteries. On the other hand, the continued rise of the pulmonary pressure at a later stage, while the systemic pressure is rapidly falling, can be explained only by the action of the pulmonary vaso-constrictors.

[2] See Dr. M. Foster's Physiology, fifth edition, p. 618.

[3] It has been proved by experiment that in the dog speedy arrest of the heart's action is induced by ligature of one of the larger branches of the coronary artery. Lectures on General Pathology, Cohnheim : New Sydenham Society, vol. i., p. 528.

effect of contraction of the systemic arterioles is a fall of pressure in the systemic veins, no such fall of pressure has been observed to occur in the pulmonary veins when the increased blood pressure in the pulmonary artery and anæmia of the capillaries indicate that the vaso-constrictors of the lungs are impeding the onward flow of the blood. As there is a direct relation between the anæmia of the minute vessels of the lungs and the collapse of those organs when the chest is opened, and as the anæmia is the result of contraction of the pulmonary arterioles, it is obvious that the greater the resistance resulting from this arterial contraction the greater will be the pressure to which the pulmonary veins will be subjected by the elastic tissue of the collapsed lungs.]

With reference to the innervation of the pulmonary vessels, Drs. Bradford and Dean have proved not only the existence of pulmonary vaso-motor nerves, but also that they leave the spinal cord higher up than the systemic vaso-motor nerves.[1] True, these authors remark that "it is probable that the pulmonary vaso-motor mechanism is but poorly developed compared with that regulating the systemic arteries." It would, however, be a strange and incredible physiological anomaly if the vessels of an organ through which the entire blood of the body has constantly to pass had not the same regulating and resisting power, compared with the force of the right ventricle, as that possessed by the systemic arterioles.

Mr. Martin has found, by introducing a manometer into a branch of the pulmonary artery of a moderate-sized cat, while the remaining branches are suddenly obliterated, that the blood pressure in the pulmonary artery rose from 17 mm. of mercury to 36 mm. But neither this nor any other experiment which has been hitherto devised can accurately measure the resisting power of the pulmonary arterioles or the actual force of the right ventricle, for the obvious reason that the arrest or great diminution of the pul-

[1] Proc. Roy. Soc., vol. xliv., No. 277.

monary circulation enfeebles the muscular walls of the heart by cutting off the blood-supply through the coronary arteries. Mr. Martin has also found that during the last stage of asphyxia the pressure in the pulmonary artery is nearly doubled, while that in the carotid is rapidly falling. That the pulmonary arterial pressure is somewhat less in the last stage of asphyxia than when the artery is suddenly and mechanically compressed seems to be explained by the fact that while in the latter case the blood in the coronary vessels is arterial, in the former the muscular walls of the heart have been weakened by the gradually increasing venosity of their diminished blood-supply. Indirect though weighty evidence in support of the theory that the anæmia of the pulmonary capillaries, with distension of the right cavities of the heart, in the last stage of asphyxia is the result of extreme constriction of the pulmonary arterioles is afforded by the well-known fact that immediately after the readmission of air to the lungs there is a great and instantaneous increase of the systemic arterial blood-pressure; the most probable explanation of the phenomena being that the reintroduction of air into the lungs causes, through the vaso-motor nerves and centre, relaxation of the pulmonary arterioles, as a result of which the blood which had accumulated in the trunks of the pulmonary artery and in the right cavities of the heart is forced onwards, at first by the mere elastic resiliency of the distended pulmonary arteries and cardiac walls; while later

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[1] Dr. M. Foster has done me the favour to direct my attention to a paper by Professor Knoll (Der Blutdruck in der Arteria Pulmonalis bei Kannichen: Sitzungsberichte der Kaiserlichen Akademie in Wien, Band xcvii., Abtheilung 14, S. 207). In this paper Dr. Knoll describes a method of measuring the normal blood pressure in the pulmonary artery of rabbits, which is less fallacious than the processes which have more commonly been adopted. He divides the sternum and introduces a tube into the trunk of the artery without wounding the pleura. By this means the blood pressure is taken, while normal respiration continues. The author admits, however, that the opening of the mediastinum and the consequent exposure of the root of the artery to atmospheric pressure may be a source of error.

the vital contractility of the heart is increased, partly by its lessened distension, and partly by an increased supply of oxygenised arterial blood.

[If the arrest of the circulation were the result of paralysis of the heart's walls by the circulation of venous blood, the arrest would be permanent and irremediable, for this theory assumes that, in order to renew the heart's beats, aerated blood must reach the coronary vessels, an event which could not be brought about without the previous restoration of the heart's action.]

I have before stated that the extreme anæmia of the pulmonary capillaries in the last stage of asphyxia and the rapid emptying of the left cavities of the heart, while the right are becoming greatly distended, are facts inconsistent with the theory which assumes that the distension of the right cavities of the heart is a result of backward pressure from the left, consequent on systemic arterial obstruction; and, on the other hand, the *instantaneous* renewal of the circulation by the readmission of air into the lungs is not to be explained by the theory of the previous arrest being caused by cardiac paralysis consequent on the circulation of venous blood.

Conclusions relating to Asphyxia.

That the immediate cause of death from asphyxia is the arrest of the pulmonary circulation appears to be proved by the following facts:—1. When the chest of an animal is opened immediately after death caused by ligature on the trachea, the right cavities of the heart are found enormously distended, while the left are comparatively empty. 2. When the heart of an animal is exposed during the progress of asphyxia the right cavities are seen to become distended, while the left cavities, which had been previously gorged, are found to be collapsed and comparatively empty. 3. In the last stage of asphyxia there is a continuous increase of pressure in the pulmonary artery, while the systemic arterial pressure is falling. 4. That the arrest of the circulation through the lungs is due to contraction of the pulmonary

arterioles appears to be proved by the influence of agents which are known to paralyse the arterioles—e.g., nitrite of amyl, atropine, and an excessive dose of curare, the effect of which is that deprivation of air is unattended by distension of the right cavities of the heart, and other evidence of obstructed pulmonary circulation, the life of the animal is prolonged for several minutes, and death ultimately results from the toxic action of venous blood upon the cardiac and nervous tissues.

[5. It is an acknowledged fact that these paralysing agents act alike upon the systemic and the pulmonary arterioles, but the successive phenomena of asphyxia are absolutely inconsistent with the idea that the distension of the right side of the heart is a result of systemic arterial obstruction acting backwards through the left cavities of the heart and the lungs.]

The Anæsthetic Action of Nitrogen alone, or with a small proportion of Oxygen.

I have elsewhere stated that the phenomena which result from the inhalation of nitrous oxide as an anæsthetic by human beings are strictly analogous with those observed during the early stages of asphyxia.[1] While some writers maintain that the anæsthetic action of nitrous oxide is due to its preventing access of free oxygen to the system, others believe that it has a "specific anæsthetic action." It occurred to me that some light might be thrown upon this subject by the administration of pure nitrogen. Accordingly I obtained from the Scotch and Irish Oxygen Company of Glasgow a cylinder containing 100 cubic feet of compressed nitrogen, in which the proportion of oxygen present was only 0·5 per cent. by volume, with 0·3 per cent. of CO_2. As a preliminary trial, Mr. F. W. Braine was good enough to administer this gas in five instances to members of the staff of King's College, who volunteered to submit to the experiments. The result was in each case the production of complete anæsthesia and of general phenomena precisely

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[1] Essay on Asphyxia, p 30.

similar to those observed from the inhalation of nitrous oxide. Encouraged by these results, Mr. Braine felt justified in administering the gas to patients at the Dental Hospital for anæsthetic purposes. Nine patients took the gas. In every case the result was the production of complete anæsthesia, with general phenomena precisely similar to those observed during nitrous oxide inhalation. The pulse was first full and throbbing, then feeble. In the advanced stage the respiration was deep and rapid, and there was lividity of the surface; the pupils were dilated, and there was more or less jactitation of the limbs. The only difference, in the opinion of some of those present, being that the anæsthesia was less rapidly produced, and somewhat less durable, than that from nitrous oxide, though in each case the tooth was extracted without pain.

On a subsequent occasion the same gas was administered by Dr. Frederic Hewitt at the Dental Hospital. As before, nine patients took the gas. The maximum period required to produce anæsthesia was 70 seconds, the minimum 50 seconds, and the mean time 58·3 seconds. In one case two teeth were extracted without pain. In one case only was pain experienced, and in that case, the tooth having been broken up and not extracted, the patient said she felt a "smashing up." Having on several occasions witnessed the administration by Dr. Hewitt of nitrous oxide mixed with 10 per cent. by volume of oxygen, with the result of producing anæsthesia without lividity or jactitation, I determined to try a mixture of nitrogen with a small proportion of oxygen. I therefore obtained from the company above mentioned a cylinder containing forty cubic feet of nitrogen mixed with 3 per cent. by volume of oxygen, and a second cylinder equally charged with a mixture of nitrogen with 5 per cent. by volume of oxygen. These gases were administered by Dr. Hewitt to patients at the Dental Hospital with the following results:—In the case of the 3 per cent. gas, which was given to five patients, the time required to produce anæsthesia varied from 60 to 75 seconds, the average time being 67·5 seconds. In each case the tooth was extracted without pain, the

duration of anæsthesia being somewhat longer than with pure nitrogen. In each case there was lividity, dilatation of pupils, and more or less jactitation. On the same day Dr. Hewitt gave nitrogen with 5 per cent. oxygen to four patients. With this mixture the time required for the production of anæsthesia ranged from 75 to 95 seconds, the average being 87·5 seconds. In each case there was complete anæsthesia, during which one patient had three molars extracted, and although she said she "felt the two last," the sensation appears to have been that of a pull, and not of acute pain. In all of these four cases there was slight lividity before the face-piece was removed, but in only one case was there slight jactitation of the limbs. The other three patients were perfectly quiescent. The experiments here recorded suffice to prove that nitrogen, pure or mixed with a small proportion of oxygen, is as complete and apparently as safe an anæsthetic as nitrous oxide. It is to be hoped that those who are engaged in the administration of anæsthetic gases will investigate this interesting subject further, with a view to ascertain whether atmospheric air, partially deprived of its oxygen, may be advantageously substituted as an anæsthetic for nitrous oxide. For the information of those who may be disposed to enter upon such an investigation, I may state that Brin's Oxygen Company (69, Horseferry-road, Westminster) are prepared to supply nitrogen containing from 4 to 7 per cent. of oxygen at the same rates as they now supply pure oxygen—viz., in quantities of twenty cubic feet or less at 4*d.* per cubic foot, of twenty to sixty cubic feet at 3*d.* per cubic foot, and of over sixty cubic feet at 2½*d* per cubic foot, in their own cylinders. Below 4 per cent. of oxygen, nitrogen could be supplied only by special arrangement, and probably at increased cost. As before stated, I obtained the gas employed in my experiments from a Glasgow firm, and its cost was therefore increased by the expense of carriage from Scotland.

Savile-row, W.